

University of Groningen

The role of viruses in marine phytoplankton mortality

Baudoux, Anne-Claire

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version

Publisher's PDF, also known as Version of record

Publication date:

2007

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

Baudoux, A-C. (2007). *The role of viruses in marine phytoplankton mortality*. [Thesis fully internal (DIV), University of Groningen]. [s.n.].

Copyright

Other than for strictly personal use, it is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), unless the work is under an open content license (like Creative Commons).

The publication may also be distributed here under the terms of Article 25fa of the Dutch Copyright Act, indicated by the "Taverne" license. More information can be found on the University of Groningen website: <https://www.rug.nl/library/open-access/self-archiving-pure/taverne-amendment>.

Take-down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

Downloaded from the University of Groningen/UMCG research database (Pure): <http://www.rug.nl/research/portal>. For technical reasons the number of authors shown on this cover page is limited to 10 maximum.

Summary

Over the last two decades, evidence has accumulated that viruses may regulate phytoplankton population dynamics. All the major classes of phytoplankton are infected by viruses. The increasing number of algal viruses brought into culture indicates high viral diversity. Observations of virally infected cells using transmission electron microscopy furthermore suggest that viruses can account for significant phytoplankton mortality. In spite of this awareness many aspect of virally mediated mortality are poorly understood and the relative impact of viral lysis as compared to other phytoplankton loss factors (e.g. grazing) is essential unknown. Furthermore, the extent of viral lysis in environments with contrasting trophic status is far from complete. This lack of knowledge constrains our understanding of the global significance of virally mediated mortality.

The partitioning of phytoplankton mortality into cell lysis and grazing is important because these loss factors influence the structure and functioning of the basis of marine food webs in different ways. While grazing basically channels phytoplankton biomass to the higher trophic levels, viral lysis shunts cellular material to the pool of dissolved organic matter (DOM) which is subsequently regenerated by bacteria. In this thesis, we have examined the role of algal viruses as compared to microzooplankton for phytoplankton mortality in environments with contrasting trophic status (eutrophic vs. oligotrophic). Details are given on the viruses infecting the bloom forming species *Phaeocystis globosa* and on the role of irradiance in regulating virus-algal host interactions.

In the eutrophic waters of the southern North Sea, *Phaeocystis globosa* typically develops dense spring blooms including flagellated cells (5-7 μm) and colonies (up to 1-2 cm). The monitoring of two consecutive spring blooms (2003 and 2004) revealed that viruses actively contributed to the demise of *P. globosa* single cells (Chapter 2). Viral lysis was the major cause of total cell lysis with rates up to 0.35 d^{-1} and even prevailed over microzooplankton grazing rates at the end of the blooms. The abundance of putative *P. globosa* virus (PgV) increased during the development of the bloom, concomitantly with the increase in virally induced mortality of *P. globosa* cells. Our results, furthermore, showed that the increase in abundance of infective PgVs was delayed as compared to total putative PgVs and viral lysis. Interestingly, this delay in infective PgVs coincided with the presence of transparent exopolymeric particles (TEP), which are generated when colonies disrupt. Because viruses can adsorb to TEP, the fraction of infective PgVs available for successful infection may be strongly reduced. This first

simultaneous estimate of viral lysis and microzooplankton grazing in the field showed that viruses are important loss agents for *P. globosa* single cells during natural blooms, resulting in virally induced mortality rates of *P. globosa* comparable to microzooplankton grazing.

Twelve viruses specifically infecting *P. globosa* (PgV) were isolated from the southern North Sea and partially characterized (Chapter 3). All PgV isolates shared common phenotypic features with other algal viruses belonging to the family Phycodnaviridae. An earlier study investigating the sequence of the conservative DNA polymerase (pol) gene also showed that the PgVs formed a closely related monophyletic group within the family Phycodnaviridae. Despite this close genetic relatedness, we could discriminate four groups of PgV based on phenotypic characteristics. Two main groups were distinguished based on particle size (150 vs. 100 nm), genome size (466 vs. 177 kb) and structural protein composition. The lytic cycle showed a latent period of 10 h for one group, but for the other group, two different latent periods for PgV (12 h vs. 16 h) were recorded. Host specificity and temperature sensitivity finally defined a fourth group. The 4 distinct types of *P. globosa* viruses described in this study were collected within a year from the southern coastal North Sea. Interestingly, different PgV groups were found to co-occur in the same water sample. The coexistence of viruses infecting different strains within a host species might indicate that viral infection is not only regulated by the overall *P. globosa* abundance but also by clonal composition of host population. Our results also indicate that co-occurring PgV groups may be competing for the same specific host strain. The outcome of these competitive interactions may influence the diversity of natural PgV population. In summary, our results illustrate that viral infection can play an important role not only in *P. globosa* population dynamics but also in the diversity of both host and virus community.

Two other field studies executed in the subtropical northeastern (NE) Atlantic Ocean during autumn (Chapter 4) and the North Sea during summer (Chapter 5) revealed that viruses may not only substantially contribute to the mortality of phytoplankton in eutrophic waters, but also in oligotrophic environments. The magnitude of viral lysis varied widely among phytoplankton groups as well as location. Our data suggest depth-variability of the extent of viral lysis of phytoplankton, but this needs further testing.

In the subtropical NE Atlantic Ocean, viral lysis and microzooplankton grazing rates of four specific picophytoplankton groups (2 size classes of picoeukaryotes, and the cyanobacteria *Synechococcus* and *Prochlorococcus*) were determined in the deep chlorophyll maximum (DCM). The DCM was chosen because algal abundance in the overlying surface water was too low for proper analysis using this method. Viral lysis, with rates up to 0.8 d^{-1} , tightly controlled the smallest picoeukaryotic group. Interestingly, these high viral lysis rates ($0.5 - 0.8\text{ d}^{-1}$) positively related to putative algal virus abundance and concurred with the highest host growth rates ($0.4 - 0.9\text{ d}^{-1}$). In comparison, the numerically dominating cyanobacteria *Synechococcus* and *Prochlorococcus* experienced no relevant loss due to viral lysis (on average 0.02 d^{-1}).

Microzooplankton grazing ($0.1 - 0.2 \text{ d}^{-1}$) appeared to be the main loss factor for *Synechococcus*, but neither viruses nor microzooplankton ($0 - 0.1 \text{ d}^{-1}$) seem to control *Prochlorococcus*. These relatively low loss rates in combination with moderate growth rates (on average 0.4 d^{-1}) may explain the numerical dominance of *Prochlorococcus* (60 - 94% of total abundance) in the studied area. Overall, phytoplankton viral lysis led to a considerable carbon release ($0.1 - 0.3 \mu\text{g C L}^{-1} \text{ d}^{-1}$), which corresponded to an average of 21% of the total carbon biomass produced by picophytoplankton in the DCM. Because the DCM represents a specific layer of the euphotic zone, these results cannot extrapolated to the entire euphotic layer. Nevertheless, our data suggest that viruses can impact a greater fraction of phytoplankton carbon production than previously assumed.

The significance of viruses as mortality agents for different picophytoplankton groups (three size classes of picoeukaryotes and *Synechococcus*) was also investigated across different regions of the seasonally (during summer) oligotrophic North Sea. Viral lysis and microzooplankton grazing were assessed in the surface waters where sizeable phytoplankton populations could be detected. For practical reasons, only one of the stations was sampled at both the surface layer and the DCM. The extent of virally induced mortality rates varied across the studied area. The highest rates of viral lysis were found for specific picoeukaryotic groups in the DCM as well as in surface waters of a Norwegian coastal station. At the three other stations (located in the offshore surface waters of the North Sea) rates of viral lysis were insignificant. Conversely to viruses, microzooplankton actively grazed upon picoeukaryotes (on average 0.3 d^{-1}) across the studied area, whereas grazing on *Synechococcus* appeared to be restricted to the Norwegian coastal station. Microzooplankton constituted the main loss factor in the North Sea during summer, consuming on average 40% of the carbon produced by picophytoplankton. By comparison, viruses induced a daily release of picophytoplankton carbon production ranging between 0 to 32% (average 8%). This study suggests that the partitioning of algal mortality into viral lysis and grazing varied widely among different regions of the North Sea during summer.

The observed differences in the magnitude of viral lysis rates in Chapters 4 and 5 led to hypothesize that the variable underwater light intensity may be involved in the regulation of successful viral infection and, thus, the significance of virally mediated algal mortality. We speculate that the high solar radiation in the surface waters might have reduced (or even prevented) viral infection of picophytoplankton and/or affected the kinetics of viral growth. Although not implicitly formulated in Chapter 2, *Phaeocystis globosa* also experiences large variation in irradiance in the turbulent, turbid coastal waters during spring. Information on how different light intensities affect virus-algal host interactions is largely lacking therefore, we investigated this issue in more detail (Chapter 6). A laboratory experiment conducted with *P. globosa*, as representative of phytoplankton thriving in eutrophic waters, and *Micromonas pusilla*, as representative of picophytoplankton abundant under oligotrophic conditions, revealed that irradiance level (0, 25, 100, and $250 \mu\text{mol photons m}^{-2} \text{ s}^{-1}$) species-specifically affected viral lysis. Both low and high irradiances (25 and $250 \mu\text{mol photons m}^{-2} \text{ s}^{-1}$) prolonged the viral latent period and/or reduced burst size of PgV. Hence, the occurrence of such light

intensities in nature may lead to a reduced encounter rate between virus and host (because of reduced PgV abundance) which, in turn, can give the opportunity for *P. globosa* to flourish. In contrast, the interactions between *M. pusilla* and MpV were unaffected by the different irradiance levels, but darkness inhibited MpV replication. Considering that the picoeukaryote *M. pusilla* and its viruses are abundant in oligotrophic waters, these results support the finding of high rates of viral lysis in light attenuated environments (as observed in Chapters 4 and 5). However, high irradiance might not always lead to reduced viral infection in surface picophytoplankton as initially hypothesized. Enhanced UV radiation might be another factor reducing the impact of viruses in surface phytoplankton population. Overall, this study emphasized the potential of solar radiation as a factor influencing virally mediated mortality.

The investigations presented in this thesis shed new light on the importance of marine viruses as drivers of phytoplankton mortality. Our results clearly show that viruses, next to microzooplankton, can be significance mortality agents for phytoplankton across ecosystems with contrasting trophic status. Hence, viral lysis of phytoplankton may substantially influence the nutrient cycling in the ocean. Last but not least, solar radiation may be a relevant environmental factor not only affecting the growth of phytoplankton, but also virally mediated mortality. Based on these studies, it appears essential to perform more targeted studies of ecologically relevant phytoplankton groups, of virus-host interactions among the different strains of viruses and hosts, and of underlying factors influencing the extent of viral lysis. Only then will we be able to obtain a better understanding of the role of viruses in marine phytoplankton mortality and within biogeochemical processes.